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EXAMINATIONS.

By A. Primrose, C.B., M.B., F.R.C.S.

At the close of the session a compulsory "stock-taking" is carried on by the University examiners to determine the progress made by individual undergraduates in their respective years. We hear a great deal regarding the evils of the examination system, and yet we could not determine the standing of students by any other means. We must therefore perpetuate this series of annual tests. Among the evils of the examination system may be cited the tendency for the student to "cram" and thus one may succeed in reaching the necessary standard by means which are unworthy and harmful. The ability to cram successfully varies greatly among students. It is mere memory work that tells in the cramming system and so it is possible for a student to "know and not understand." It is the reasoning power that forms the greatest asset of the student in any branch of learning and more particularly is this the case in scientific training such as is required in the profession of medicine. Given the necessary mental equipment, it is the hard, conscientious work from day to day throughout the session that produces the best student and, one may add, that assures his success in the final examinations. The teacher who meets his students from day to day in personal contact can usually predict, with great accuracy, the fate which awaits the candidate in the examination ordeal. There may be a few doubtful cases and, it would appear, that the main function of an examination is to determine whether or not these weak candidates should get their standing. Another possible value of examinations is to determine comparative attainment for honours. While therefore the present system has certain advantages, there is nevertheless something to be said for the entire elimination of the annual examination. It would appear desirable, if it were possible, to devise some other method of determining the students' fitness to pass on from year to year in the curriculum of study.

One cannot consider the examination problem from the standpoint of the student alone. It is difficult to get an ideal examiner. There is often a temptation to try to find out what a student does not know rather than to determine what he does know. Let me quote from an author who wrote more than a century ago:

"Examinations are formidable, even to the best prepared, for the greatest fool may ask more than the wisest man can answer." (Chas. Caleb Colton.)

There can be no doubt of the fact examinations are unsatisfactory tests whether viewed from the standpoint of the candidate or the instructor, and yet they appear in the meantime to be a necessary evil. Let me close these observations on examinations by a quotation from Osler.

"Perfect happiness for student and teacher will come with the abolition of examinations, which are stumbling-blocks and rocks of offense in the pathway of the true student."

EDITOR, 1928-29.

Mr. R. M. McKeown, M.A., has accepted the appointment of Editor-in-Chief of this Journal for the coming season. Under his able direction the Journal is sure to flourish and be a still more integral part of the Undergraduate body. We wish him and his associates every success.

* * *

The Editoral Staff takes this opportunity of expressing its appreciation to those who have contributed to the various issues, and thus in giving their time have reaped a great reward in making the existence of the Journal possible. We also wish to thank our advisers in the Faculty of Medicine for their co-operation and advice.

* * *

Professor J. J. R. MacLeod has accepted the appointment of Professor and head of the Department of Physiology in the University of Aberdeen. Congratulations and best wishes.

SKIN ERUPTIONS DUE TO FUNGI.

Hamnett A. Dixon, M.B. (Tor.)

To make my notes as brief as possible, I shall limit myself to a description of the more recently recognised fungus infection, i.e., Epidermophytosis.

Ten years ago, the infection was uncommon in Canada. Dermatologists were not familiar with its characteristics. From the year 1918 to 1924 there has been a steady increase in the prevalence of the disease. Previous to the war the infection was common in tropical countries. It was common among the troops in France, in the East, and in England. With the return of so many to civil life the disease has become widespread. Persons of all ages are susceptible. It is of special interest to know that we see the disease in children more frequently each year.

Epidermophytosis is caused by a mould. In what manner the infection is contracted is difficult to answer accurately, but experience shows that the plant gains entrance to the skin in many ways. Among the most common sources of infection are woollen socks, and to a lesser extent woollen gloves. Leather is a source of infection. Cases are seen due to baseball gloves, handles of golf clubs, tennis-racket handles, and even the strap of a wrist watch. Suspensory bandages and athletic clothing worn many times, soaked with perspiration and not properly washed are common sources. The floors of shower baths have proved troublesome. Outbreaks are found in boarding schools, in golf clubs and various places where many people make use of the same shower bath.

It has been found that twice as many males as females are infected. This may be due to the fact that men take more athletic exercise and therefore come more in contact with wool, leather and shower baths.

Epidermophytosis is capable of producing many varieties of lesion and of attacking almost any part of the skin. Also, the toxins may be absorbed and produce distal lesions corresponding to the tuberculides.

Vesicles or small serum blisters form the most common type of eruption, and as primary lesions, seem to be limited to the fingers and palms, to the toes and soles. The vesicles frequently have a dark steel blue colour, a sign which should always make one suspect epidermophytosis. The vesicles prefer the interdigital surface, but when more numerous they spread to the flexor sur-

faces. The eruptions are more common in the warmer months. They may be present without subjective symptoms but the majority of patients complain of itching and burning of a most severe character.

The scaling form is the second most common type. The scales are seen on the sides of the fingers and toes or on the palms and soles. When examining the feet it is necessary to separate the toes and examine, especially in the fourth interspace or at the junction of the toes and the ball of the foot.

The third most common variety of lesion is that mild inflammatory type producing a reddish discolouration of the skin with a raised margin covered by a fine gray scale. It appears in the groin and may extend down the thighs or backward over the perineum. It does not disappear by clearing in the centre as in ordinary ringworm. The axillae may be infected As a rule the lesion remains limited to be bottom of the axilla, but may extend downward over the side of the chest and abdomen. The scaly patches may appear under the breasts, about the umbilicus, and occasionally over the larger flat surfaces of the chest and abdomen.

Other lesions seen are maceration of the skin between the toes and fingers. It appears as white sodden tissue of varying degrees of thickness. Again, one may find areas of callous over the heel and the ball of the foot. The callous is generally free from subjective symptoms except where deep fissures appear and then walking is painful.

Nail changes are not uncommon. They occur as thickening, with change in colour, vertical ridges, crumbling and separation of the nail plate from its bed.

Where the diagnosis cannot be made from the clinical picture it is necessary to look for the fungus in small pieces of the affected skin. The specimen most likely to show the parasite is the steel coloured blister or the brownish scale which results from the drying of a blister. The specimen is mounted in 10 per cent. potassium hydroxide solution and examined with the high dry lens. The fungus will be seen running between the epidermal cells as a chain of oval or rectangular beads. When the organism cannot be demonstrated it is necessary to cultivate on artificial media. The medium most suitable is one made with maltose, peptone, agar and water (Sabouraud's). The maltose inhibits the growth of contaminating staphylococcides. The parasite grows best at a temperature corresponding to that of the body surface, i.e., at laboratory room temperature. It thrives in shaded light or in a closed cupboard. If grown in an incubator growth is too rapid and the characteristic morphology is lost.

Growth is slower than that of bacteria, the earliest signs appeared as a yellowish white tuft about the margin of the scale, seven days after planting. After two weeks the colony showed a crater-like centre with divergent furrows running from the centre to the margin.

Treatment: The patient must be warned against wearing wool, silk or leather next to the skin. Only cotton material which may be boiled should be permitted. Shower baths must be forbidden unless the affected feet are covered with cotton socks or paper slippers. Local treatment has been disappointing. The lesion clears readily, but is apt to recur periodically, in warm weather, when the spores or seeds of the plant develop. It is diffiicult to destroy the spores without damaging the skin. Success is only obtained by energetically treating after all lesions have cleared.

In the acute stage all small blisters should be opened and the moist surfaces touched with tincture of iodine once a day. After blisters have ceased forming, the parts should be washed daily with tincture of green soap, hot water and a soft brush. Following this, paint the affected surface with a 2 per cent. aqueous solution of mercur-

ochrome—220. This is best applied with a sterile swab at first once a day and later twice a day. Mercurochrome has a drying effect on the tissues and it is probably due to this effect that the infection is destroyed. Plants require moisture to thrive.

In conclusion: Fungus infection usually refers to bald scaly patches on the head or scaly rings about the neck, fact and arms. By the demonstration of epidermophyton and related fungi we prove the existence of various skin eruptions with varying symptoms and resembling but slightly the common ringworm.

PARACENTESIS THORACIS AND PARACENTESIS ABDOMINIS.

(By W. V. Watson, M.B.)

The withdrawal of fluid from the chest by means of a trocar and cannula or aspirating needle is termed paracentesis thoracis.

The best position for the patient is the sitting, and the site of puncture the seventh interspace in the midaxillary line. For small quantities of fluid, the site will depend upon the extent and position of the fluid.

The preparation of the skin is carried out by painting with Tincture of Iodine on the clean skin and if one is desirous of getting a clearer impression of landmarks, Alcohol may be used to clear the Iodine stain.

Local anaesthesia is produced by a 1% solution of Novocaine. This substance is used in preference to Cocaine because it is possible to have it repeatedly sterilized without loss of potency and it is somewhat less toxic. The injection is done with a 2 c.c. hypodermic syringe. The skin is first anaesthetized by producing a wheal which will have a blanched appearance, then the solution may be carried into the deeper structures.

An aspirating needle is more commonly used than trocar and cannula in removing fluid from the chest. Short needles are satisfactory when the fluid is present in large quantities, but for the smaller collections longer needles are frequently necessary.

One of three arrangements of apparatus will suit any particular case for paracentesis. The first and probably the simplest plan is suitable when the fluid will flow freely through the needle. It is necessary to have a length of rubber tubing filled with sterile normal saline and clamped at both ends, it must be long enough to reach a receptacle below the level of the patient. With the tube attached to the needle the puncture is done and when the needle reaches the fluid the clamps are released allowing it to run freely.

The second method is suitable in those cases where the fluid is difficult to find or when it contains particles of fibrinous exudate which tend to plug the needle. An all-glass syringe is used to which is attached the needle by means of a three-way valve. This arrangement allows one to fill the syringe from the chest and by turning the valve it may be emptied into a receptacle without detaching.

In the third plan an aspirating bottle is used and the fluid is removed by means of a negative pressure produced in the bottle with an aspirating pump. The needle is connected with the bottle by means of high pressure rubber tubing, i.e., tubing made with a thick, firm wall. The puncture is done with the needle attached to the tubing, and when the fluid is reached the tap to the bottle is opened. In producing a negative pressure in the bottle it must be remembered that the pump has two nozzles, one producing a negative pressure, while the other is used to produce a positive pressure. It is well to

try out the aspirating bottle with a little sterile water before doing the puncture.

This third method is most suitable in cases with moderate to large quantities of a transudate or a purulent fluid if it does not contain masses of fibrin which tend to obstruct the flow even with the negative pressure of this equipment.

In doing the puncture care must be taken to avoid the intercostal vessels and nerve. Also, in cases with small amount of fluid situated rather low, one must have due regard for the diaphragm and the structures beneath it. It is a wise rule to remove fluid slowly and should pain or cough occur, the needle should be withdrawn slightly so that it will avoid irritation. Violent cough, rapid pulse or syncope is a signal to stop the procedure.

The paracentesis is followed by a simple dressing of the puncture site with a wisp of cotton saturated with flexile collodion.

By means of paracentesis abdominis fluid is removed from the peritoneal cavity with a trocar and cannula. It may be repeated many times with practically no danger to the patient. It is done in the sitting position or with the patient lying in bed.

The cleansed skin of the lower abdomen is prepared with Tincture of Iodine and Alcohol. One of two sites may be used, the first in the mid-line half way between the umbilicus and pubis or the second just outside the rectus muscle on a line midway between the umbilicus and the anterior superior iliac spine. Adhesions from punctures necessitate a change of site. Local anaesthesia with novocaine is carried out as for paracentesis of chest. A small incision is made through the skin to allow the trocar and cannula through more easily and also to avoid the carrying through any skin infection.

The trocar and cannula for this purpose is designed so that a piece of tubing may be attached without removing the trocar. The tubing must be of sufficient length to reach a container below the level of the patient as the fluid is generally removed by gravity. In cases where the fluid is limited by adhesions an obturator is provided to replace the trocar so that the search for fluid will not injure the viscera. An abdominal binder is provided to produce pressure temporarily, if necessary, during the procedure and to apply in the usual way after the fluid is withdrawn.

The puncture is done by holding the trocar and cannula firmly in the hand and it is kept in place until all the fluid that can be reached is run out. It is then removed and a collodion dressing applied and the binder fixed. If the incision is made only large enough to allow the cannula through it may not require a suture, but if larger it must be closed before applying the dressing.

While it is advantageous to do paracentesis in hospital, it is possible to carry it out efficiently in the patient's home in clean surroundings.

The Graduating Class of 1929 offer a prize of Ten Dollars to the author of the "skit" chosen to be used by this class on Daffodyl Night of the coming year. Manuscripts are to be given to any of the class executives or to be left at the offices of the Medical Society during the Fall term.

THE RESPIRATION IN ANAESTHESIA.

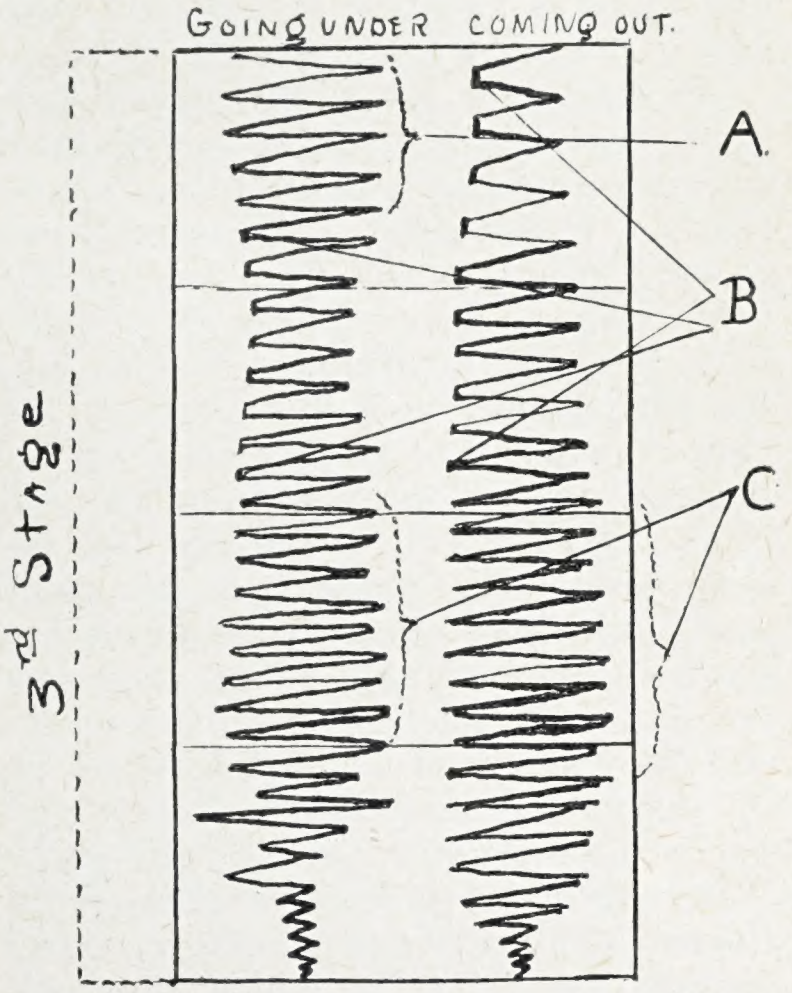
(By H. J. Shields, B.A., M.B.)

To the experienced anaesthetist, the administration of an uncomplicated anaesthetic, particularly after surgical anaesthesia has been obtained, is usually an easy matter. If giving ether, at certain intervals a quantity of this anaesthetic is dropped upon the mask. Different types of patients require considerably different amounts of anaesthetic, or that amount required cannot be governed by the clock, so to speak. In spite of this factor, to the man who has given many anaesthetics, the maintenance of a smooth anaesthesia is in most cases a simple procedure. Almost unconsciously he will reach out for the ether dropper and administer more of the anaesthetic. Frequently he will do this without any realization whatever of having done so. In other words his actions are more or less automatic. Unquestionably his administration of anaesthetic at certain intervals is a response to some sign or signs exhibited by the patient. What then is the nature of this instinct?

As has been stated the experienced anaesthetist without inspecting his patient for any sign, at a certain moment will add anaesthetic. Also he can and does maintain anaesthesia in the dark room, for considerable lengths of time if necessary, without experiencing any uneasiness. He can walk into an operating room and almost instantly state the depth at which anaesthesia is being maintained without looking at the patient. Therefore, he is being guided, not so much by what he sees as by what he hears. However, it is the experience of most administrators that it is difficult to maintain smooth anaesthesia in noisy surroundings. The eye signs are about the only ones of any value as a guide to the depth of anaesthesia, which may be obtained by inspection of the patient. There are other conditions such as color of the patient of which the anaesthetist must note, but they are of small value in determining the depth of anaesthesia. It is obvious that it is impossible to keep these eye signs under observation every moment of the anaesthesia, and therefore they cannot be responsible for the instinct which has been mentioned and which undoubtedly is present. It follows, then, that it is the breathing of the patient, the only audible sign of anaesthesia always present, to which the administrator is responding. An analysis of this breathing of anaesthesia may throw some light on this question.

In the first two stages of anaesthesia there can be no classification of the breathing since this breathing is influenced by many conditions. In the first stage the patient is conscious and the breathing therefore under voluntary control. In the excitement stage the breathing will vary with the nature of excitement present. With the advent of the third stage, however, the breathing becomes regular and distinctly audible. Indeed this is the one valuable sign of beginning surgical anaesthesia. It differs from normal breathing in three respects, an increase in rate, an increase in depth, and what is more important, a change in the respiratory ratio. Now, this third stage of anaesthesia is broad in comparison with the other stages with the signs varying at different levels within that stage Considering at this time the respiration only we find, then, that with the advent of surgical anaesthesia no matter what the induction is like or how brought about, in most cases the breathing becomes what is known as automatic breathing, that is, with inspiration and expiration equal in length with the pauses at the end of each also equal and practically negligible. If it is not of this type it will closely approach it.

When sedatives have been employed this latter type will usually be present. There is something about automatic breathing which makes one instinctively feel that the patient is deeply unconscious and this is probably why so often beginners ease up on the anaesthetic at this point, only to find in a moment or two that their anaesthesia is decidedly light. With more experience, however, one neglects the presence at this time of automatic breathing, although, as will be seen, it is the breathing one expects to find in mid-third stage. As more anaesthetic is given, in the course of two or three minutes the respiration changes and becomes the type that more accurately represents this early third stage. That is, it becomes slower with somewhat diminished depth and therefore a little less audible, with a pause at the end of expiration. As anaesthesia is deepened this pause gradually becomes shorter, the respiratory rate and excursion greater until the automatic rhythm is again established. This occurs at about the middle of the third stage; in fact this, with the fixing of the eye ball mark off the middle of the stage. It is seldom that anaesthesia need be pushed beyond this point. When it is, however, the rate quickens, the breathing becomes deep and noisy, more abdominal in type, then irregular, and finally after a series of rapid and shallow breaths respiration ceases at the end of the third stage. With the patient coming out more or less the exact reverse of this takes place until the middle of the third stage is reached when a very short pause commences at the end of expiration. As anaesthesia lightens further the pause at the end of expiration gradually increases in length, the rate becomes slower and the breathing less audible until, at the beginning of the third stage, the breathing resembles that of natural sleep Vomiting or involuntary muscular movements are likely to occur at this point. It will be seen that the respiration of the patient coming back through this stage corresponds to that going under except in the first plane where, in the latter case, with the advent of the third stage for a few moments automatic breathing or something closely approaching it is present.



A—Automatic Rhythm.
B—Pause at End of Expiration.
C—Automatic Rhythm.

When third stage anaesthesia has developed, the anaesthetist consciously or unconsciously is listening to the breathing of his patient. If the breathing becomes quieter with a decided pause at the end of expiration he will add, soon instinctively, more anaesthetic. When automatic rhythm develops, in the same way he discontinues to do so. When it comes to a final analysis the cardinal sign of anaesthesia from a practical standpoint is the length of the pause at the end of expiration. This is the factor to which most anaesthetists are continually responding whether they realize it or not and as has been stated the response soon becomes more or less automatic. After all, the respiration is a guide that is continuously present. The eye signs

will be seen at intervals only and in some operations not at all, as the eyes will be covered. Inspecting these eye signs even at frequent intervals cannot produce this automatic reaction which enables one to keep an anaesthetic at a given level, which reaction one believes to exist in any one with any great experience in anaesthesia.

It cannot be truthfully stated, perhaps, that the respiration is the most accurate guide as to the depth of anaesthesia. Frequently, with anaesthesia carried along in the first half of the third stage, a severe surgical stimulus with effect the breathing. If very light the patient may hold his breath at the end of inspiration and phonate on expiration. If not quite so light but still in the first half the breathing may become automatic, which does not mean that mid third stage anaesthesia has suddenly been brought about. It is just the patient's reaction to this stimulus and the anaesthetist is not deceived by it. Also some patients of a highly sensitive make-up will develop automatic breathing and will retain it throughout almost their entire anaesthesia irregardless of the depth at which they are carried. Again, very young children and infants do not go according to rule, infants particularly being a law unto themselves as regards respiration. Bearing these exceptions in mind one will seldom be deceived by the respiration. In these cases one must be guided by the other signs. Indeed one should take advantage of all signs of anaesthesia if one is to avoid the pitfalls awaiting careless feet. The breathing being present and registering on the consciousness of the administrator at all times makes one feel that the analysis of the breathing, even if not necessarily the most accurate, is the most important guide in anaesthesia.

To sum up, one can state that the automatic rhythm indicates mid third stage anaesthesia the exceptions being the few moments after third stage anaesthesia has just developed, during which this rhythm usually occurs; when an unusually severe surgical stimulus is exerted with the patient only lightly in this stage; and in certain sensitive individuals whose respiration is often of this type almost throughout their entire third stage. In the various levels leading up to the point where automatic rhythm develops certain features of the breathing, particularly the length of the pause at the end of expiration, gradually and constantly change as the depth of anaesthesia changes. Therefore, since the features of the breathing are constantly being carried to the consciousness of the anaesthetist, one feels these to be the factors which enable him to maintain a constant level of anaesthesia with so little mental effort. One also feels that an anaesthetist is not a capable anaesthetist until he is able to depend for guidance chiefly upon some such principles.

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CARCINOMA OF THE BREAST.

(*By E. A. Keenleyside, B.A., 2T8*).

Introduction.

The breast shares with the uterus the distinction of being the commonest site for carcinoma in the human body. About 40% of all carcinomas occur in this gland and about one half or more of the growths in the mammary gland are of the nature of carcinoma. These facts above show the great importance of such a condition and are further accentuated when we come to study the mortality rates. It is essential to recognise malignancy early. If we are to save the life of the patient by a radical operation. Inasmuch as early diagnosis is of the utmost importance, knowledge of the underlying pathological condition, including the mode of spread and location of metastases is of equal importance when we come to consider the efficient eradication of the disease. Over 100,000 die of cancer every year in the United States. Of these, about 10,000 are carcinoma of the breast and a big percentage might be averted by timely surgical treatment.

Incidence.

Cancer of the breast is a disease of the middle age, 40-55, but has been known to occur in the early twenties and even as late as 90 years of age. Females are much more commonly affected—about 80 cases to every 1 case in males. This is probably owing to the larger size of the gland in the female, its periodical functional activity, and to the greater incidence of disease of other kinds in the female breast. Women suffer most from carcinoma at or after the menopause. Eighty percent of women who are the subjects of mammary carcinoma have been married, and at least 70 percent of these have borne children. The disease is more malignant in young women.

It is out of the question here to enter into a discussion of so difficult and incomplete a subject as the etiology of carcinoma of the breast. The part played by trauma in the causation of cancer of the breast is still undecided, but it is generally believed that it plays a minor role. Heredity may also play a minor role as there appears to be something in the actual epithelium of the family which causes it to be liable to the incidence of carcinoma. Innocent tumors of the breast occasionally take on malignant characteristics. There are, however, three thoughts that are worthy to bear in mind when discussing the etiology of the disease.

1. *The first is*: The tissues concerned in hyperplasia cannot all be in the same biological state throughout the whole breast during the whole of a patient's life and even when normal they cannot all be able to respond equally and at the same time to a stimulus of hyperplasia. For hyperplasia to occur in a tissue it must be in a biological state that renders it capable of responding by growth to a hyperplastic stimulus.

2. *The second is*: Stimulus that is capable of inducing hyperplasia may not always be the same stimulus, nor applied in the same degree. Even when a stimulus is acting on tissues in different biological states, hyperplasia may result in some of the tissues and not in others. Again it is conceivable that two or more kinds of hyperplastic stimuli may be applied at the same time and may induce hyperplasia in two or more different kinds of responsive tissues.

3. *The third thought is*: It may be that none of the forms of hyperplasia are induced by a stimulus, but that hyperplasia may be caused by the in-

hibition of some controlling factor of growth.

Anatomy.

Before discussing the mode of spread and location of metastases, it is important to review the lymphatic anatomy of the parietes. The lymphatics of the skin originate in the papillae as blind ends which are the central structure of each papilla. The vessels leaving the papilla unite by groups to form less numerous vessels which pierce the deeper layers of the dermis and the underlying subcutaneous fat, running more or less vertically in respect to the skin surface, and after again uniting by groups in the subcutaneous fat, discharge into the lymphatic plexus which lies upon the deep fascia and thus to the axillary glands. The skin appears to be divided into small lymphatic areas, each probably corresponding to a vertical collecting tributary of the fascial plexus and to the little group of papillary vessels which unite to form it. The size of these primary lymphatic areas of the skin are from 1-3 to 1-2 an inch in diameter and there seems to be little communication between adjoining areas except through the medium of the fascial plexus. Thus independence of adjoining lymphatic areas of the skin is a fact of great pathological importance. Besides the small tributaries which dip down vertically into the fascial plexus from the surface of the body, there are running upwards to the deep aspect of the fascial plexus numerous vessels by means of which, it communicates with the lymphatics of the subjacent tissues. Such vessels have been frequently observed to pass to the deep fascia from the muscles and the special liability of some of the subcutaneous areas of the skeleton to cancer appears to show that the fascial plexus anastomoses in a similar way with the periosteal lymphatics wherever they approach the surface.

The deep fascial lymphatic plexus, with its network of intercommunicating channels, invests the entire body. This great plexus is divisable by the median plane of the body and by two horizontal planes passing through the clavicles and through the umbilicus respectively, into six catchment areas, three on either side draining, as the case may be, into the cervical, the axillary, or the inguinal glands. Within each area a special set of trunk lymphatics arises from the plexus and converges on the corresponding set of glands. The line or zone, separating any two adjacent areas may be called the lymphatic water parting and is anatomically a zone of narrow tortuous channels nowhere traversed by trunk lymphatics, a region consequently where the lymph stream is at is feeblest and where even very fine particles are liable to be arrested. Thus we have a system receiving above numberless fine vertical tributaries which convey the lymph from the skin and its appendages—among the latter we must include the breast. On its deep aspect the plexus receives tributaries from the subjacent tissues. From this great plexus which lies in the subcutaneous fat upon the deep fascia, the lymph is conveyed by six sets of lymphatic trunks each draining a definite area, to the cervical, the axillary or the inguinal glands.

Metastases.

It has now been established that carcinoma of the breast slowly spreads centrifugally and by continuity from its seat or origin. It proceeds by direct growth along the fine lymph vessels by what is known as permeation. The axillary lymph nodes are very soon involved and from here the cancer cells pass on to the next set of glands. They pass into the lymphatic vessels of adjoining areas and permeation results in the impregnation of the entire lymphatic system with cancer. Metastases occur in the lymph nodes, the pleura, the lungs, the dura, peritoneum, skin, liver and also the spleen and adrenal glands. If a case of carcinoma of the

breast could live long enough almost every bone in the body would be involved, but as a rule the bones distal to the knee and elbow escape except in the rarest instances. The liability of a bone to cancerous metastases increases with its proximity to the site of the primary growth. Cancer invasion of the long bones renders them very liable to pathological fracture.

Symptoms and Signs.

There are a few symptoms and signs of early carcinoma of the breast — which is quite important and it is well to bear in mind that either cancer or papilloma can be present in the breast without causing any clinical sign whatever. Therefore the presence of even one definite clinical sign is of fateful importance.

Nodularity.

1. *Nodularity*, localized to one point of a breast, or one part of a breast where nodularity is more marked than in any other part, may be present. In about 60% of cases the growth begins in the upper hemisphere of the gland and in its outer quadrant. It is a definite irregular, hard, non-sensitive lump that cannot be moved independently of the breast and shades away gradually into the adjacent breast tissue without any clearly defined line of demarcation between tumour and breast tissue. Palpation is the most valuable single method of examination for early carcinoma, but it should be done with utmost gentleness as vigorous palpation dislodges cancer cells which may be carried as emboli to cause distant metastases.

Pain and Tenderness.

2. Throughout life a certain amount of *pain* in the breasts is so common that many women regard it as a normal experience (e.g.) at puberty they experience mammary pain, repeated throughout the term of their menstrual periods; any irregularity of the pelvic visceral organs induces pain in the breast; various emotions give rise to it, etc. Thus it is difficult to attempt to elicit pain as part of a history of a pathological lesion. It is usually considered that pain and tenderness are late symptoms in carcinoma of the breast and it is rare for a patient to state spontaneously that she had noticed any particular pain before she felt the lump in the breast. On the other hand, Cheattle says that if a history be carefully taken and you make a point of asking about pain—she will often say—"Oh well, I did notice a little stabbing pain, but I did not take any notice of it." He says that this is a common experience of his and he believes that pain of this lancinating, stabbing character is often the earliest symptom of cancer of the breast. He operated on one case whose only symptom was localised stabbing pain, no lump nor nodularity could be felt, and on microscopic examination a small focus of carcinoma was discovered which was already beginning to spread into the fat surrounding the periphery gland.

Carnett of Philadelphia regards pain and tenderness as late stages of the disease and therefore their absence must never be entered as evidence against early carcinoma.

If the pain is superficial it is usually a warning that ulceration is impending. If deep seated, the presence of pain in the breast may be an ominous sign that the deep structures of the thorax have already been invaded. Pain, mild or severe, radiating down the arm on the side of the lesion will frequently be found to be due to metastatic invasion of the axillary or supraclavicular lymph nodes. Deep seated pain, sharp or dull, either in the thorax or abdomen, occurs with invasion of the serous membranes. Pain in carcinoma of the breast is most frequently due to skeletal metastases and in no other tissue or organ does breast carcinoma give rise to pain of such widespread distribution and in such widely varying forms, from the mildest to the severest, from the dullest to the most acute, excruciating

and agonizing as when it metastasizes to the skeleton.

3. *A spontaneous intermittent discharge of blood* from a nipple (i.e.) a discharge not caused by trauma. It may be due to (1) duct carcinoma. (2) carcinoma arising deep in the breast in terminal ducts and acini or (3) to papilloma.

4. *A spontaneous discharge of serum from the nipple,* may be caused by the same diseases that induce haemorrhage from the nipple and should be regarded with the same degree of seriousness.

5. *A gradually retracting nipple and cutaneous dimpling* are important diagnostic signs of a later stage. This confirmatory sign of carcinoma is elicited by gently lifting the whole breast forward while observing the tumor region for evidence of flattening or dimpling of the skin.

6. *Puckering of the skin over one breast* or any abrasion or ulceration near the nipple.

The above are the local signs and symptoms of the disease, but as it continues to spread—constitutional symptoms become prominent such as — a gradual loss of weight, repeated weak spells and a general feeling of fatigue; loss of appetite with some vomiting and as the various bones become involved we have pain and a tendency towards spontaneous fracture. A moderate degree of anaemia is also typical.

Differential Diagnosis.

In the differential diagnosis the following must be considered:

(a) Inflamatory Tumors.

1. Mastitis—(a) Acute, or
   (b) Chronic
   (with or without suppuration). Mastitis is exceedingly common.
2. Tuberculosis
3. Syphilis.
4. Actinomycosis.
   (2, 3 and 4 constitute the rarer forms of specific breast infection, resulting in tumour formation).

(b) Malignant Growths.

1. Cancer of Breast is by far the most malignant and is a single lump, hard, not confined to one section, neither painful nor tender.
2. Sarcoma occurs as a very rare event.

(c) Benign Growths.

1. *Adenomata or Fibro-adenomata*: The mass is single or multiple, sharply circumscribed, not attached to the skin or deep fascia, and the axillary glands are not enlarged.
2. Angioma
3. Myxoma
4. Lipoma
   (2, 3 and 4 are much rarer forms of benign tumours.)

(d) Cysts.

The tendency to cyst formation in tumours of the breast is very marked, and is found both in inflammatory conditions and in new growth. Cheattle has shown very conclusively that the cyst acts as a predisposing and determining cause in the initiation and growth of tumours both simple and malignant.

Treatment.

Whatever the clinical or pathological type, the proper treatment in all operable cases is radical removal of the breast in one mass with all underlying muscles, a very wide circle of fascia, all the contents of the axilla and, if necessary, of the supraclavicular triangle. No operation that fails to remove all adjacent fascia and muscles and to clear the axilla should be performed. Cancer should be regarded as a semi-emergency lesion and the patient be forced into hospital at the earliest possible moment to forestall the distant metastases which are inoperable.

Operative details are out of place here—but it should always be remembered that the disease spreads roughly in a circle from its point or origin and that the primary plane of spread is the

fascial lymphatic flexus, not the skin. One or two points in the operative technique should be noted here.

1. The diathermic knife is preferable to the scalpel—the operation can be performed as rapidly and with less bleeding and shock if used.

2. Inflammation and dissemination of cancer during an operation is a danger and must be guarded against in operative technique. Massage and manipulation may spread it and therefore the greatest gentleness in manipulating a cancerous breast, both before and during operation, must be observed.

Post Operative Treatment — includes the following:

Complications.

1. *Shock*—usually occurring 3 or 4 hours after return to bed. It is not often seen, but it should be anticipated.

Routine Treatment—1 pint of saline with 5% glucose per rectum immediately after operation. Repeat in 4 hours. Wrap patient in two blankets and place under cradle with incandescent lamp. Hypodermic of morphine if patient is restless or in pain should be given.

2. *Pain*—is not severe after the first night and is excited later on only by the early dressing which leads to movement of the arm or body.

3. *Chest Complications*—are rare and best avoided by careful preparation, especially of the upper air passages, by closing the wound tension and by avoiding tight bandaging. Patient is made to sit up in bed after the first 24 hours or less.

4. *Paralysis of Upper Arm* — occasionally occurs due to pressure on the outer cord of the brachial plexus due to excessive extension of the arm during operation.

The value of the use of x-ray or radium as a prophylatic means for recurrences following operation has yet to be proven. However, in the hopeless, inoperable cases of carcinoma of the breast, radium or x-ray treatment has made the remaining days of the patient more comfortable.

The treatment of carcinoma of the breast may be summed up in the aphorism of Dr. Finney—"Any lump in any woman's breast is better out than in."

Prognosis.

Prognosis in breast cancer is difficult to make. The disease may be fatal in 3 months or only after 40 years. If left untreated the average duration of life from the time of the first symptom to death varies from 27 mos. to 48 mos. With treatment by operation the prognosis depends considerably upon the amount of glandular and lymphatic involvement at time of operation and the degree of malignancy. Dr. Primrose gives the following statistics from his records of 448 cases.

1. Where no glandular metastases—90% recovery for 5 years.

2. Where glands slightly involved—61% recovery for 5 years.

3. Where glands markedly involved—9% recovery for 5 years.

In most acute forms with "inflammatory manifestations" the prognosis is easy. In the more chronic forms it is difficult, as it may creep about in tortuous recesses of the parietal lymplatic plexus for years—ultimately at some point or other permeation penetrates the defences of the body cavity, attacks the bones or reaches the cranial cavity. When once evidence of visceral disease has declared itself, it is rare for life to be prolonged for more than a year and a definite forecast of the future can be given.

Dr. Primrose's statistics prove that carcinoma of the breast can be eradicated and a permanent cure effected provided the operation is undertaken early.

CHRONIC ULCERATIVE COLITIS.

Ambrose G. McGhie, M.D.

"Chronic Ulcerative Colitis" has been dealt with in the literature under such names as Idiopathic Ulcerative colitis, non-specific ulcerative colitis, Asylum dysentery, innominate ulcerative colitis and chronic ulcerative colitis.

Physicians interested in chronic ulcerative colitis have in the past been divided into three groups based on their opinions as to the etiology of the disease.

(1) Those who believed it was an infectious process and advocated its management as a chronic, severe, debilitating infection.

(2) Those who believed it was metabolic in origin and searched for some underlying deficiency.

(3) Those who believed it was a surgical problem and advocated various operations.

Those who adhered to the first group and looked upon the disease as infectious were again divided among themselves as to the causative organism and nearly every organism ever found in the colon has been accused by one or another writer of being responsible for or contributing to the disease. The second group, blaming metabolic disturbances had a great deal of evidence to support its view, but not sufficient clinical results to convince the skeptical. The third group devised a great variety of surgical operations with varying degrees of success, but in the main unsatisfactory. It must be acknowledged that where there is such a wide divergence of opinion, among conscientious investigators on the etiology of a disease, the true causative agent is still unknown. We are, however, rapidly getting at the true facts of many diseases previously in this category, and to Dr. J. Arnold Bargen must be given the credit for discovering the etiology of chronic ulcerative colitis. Having seen his experimental and clinical work one is convinced that he has found the responsible organism.

The organism which Bargen isolated is described as a Gram-positive diplococcus which has a tendency to be lancet-shaped and is a little larger than a pneumococcus. It does not form capsules, shows little tendency to form in chains and has definite and characteristic cultural features. The organisms are obtained from the base of the ulcer through the proctoscope. Injecting cultures of this organism intravenously into rabbits, Bargen has produced lesions in the rabbit's colon essentially like those of ulcerative colitis in man. He has re-isolated the diplococcus from the gall-bladder, mesenteric lymph nodes and blood stream and re-injected these producing similar lesions. Probably the most striking and certainly a very important feature to the clinician, is the fact that Bargen has isolated this organism from the tonsils and from periapical abscesses of patients during acute exacerbations of the disease. Some patients have had acute exacerbations of the bowel disease after extraction of teeth and tonsillectomy. Since this is also inclined to follow acute infections of the upper respiratory tract the conclusion seems obvious that the disease is infectious and occurs periodically as a result of the release of these organisms into the blood stream.

In recent years reliance for the diagnosis has been placed chiefly on a combination of the findings, of the X-Ray and sigmoidoscope. The X-Ray shows certain characteristics such as spasm and marginal feathering in the acute stages, and stricture and *obliteration of the haustra* in the chronic. In 20 per cent. of patients the rectum alone is involved and barium enema does not give any information. The appearance of the bowel through the sigmoidoscope

varies with the degree of involvement, and the stage of the disease at the time of examination. Few examinations are made during the first attack, but at this stage there is a diffuse hyperemia of the mucosa most marked near the anus and lower rectum; the upper margin, if visible, fades out into the normal mucous membrane. Later there is cedema and thickening of the mucous membrane with hyperemia persisting. At this stage the slightest pressure by the examining instrument will break the mucosa. Following this miliary abscesses form and are situated diffusely throughout the diseased area. The abscesses are located immediately beneath the superficial layer of the mucosa and are usually about 1 m.m. in diameter. These abscesses eventually rupture, leaving miliary ulcers with a yellowish base, which bleeds easily. Remissions frequently occur at this stage and if healing takes place the mucosa remains pitted or pock marked at the site of lesions. However, healing may fail to take place after such an exacerbation and secondary infection aids the spreading of the ulcer into the surrounding mucosa. Then superficial ulcers up to 3 cm. in diameter with ragged edges are found. This is the type of lesion that causes the greatest amount of destruction to the mucous membrane.

The salient clinical feature of the disease is the frequent passage from the rectum of a discharge containing blood and pus in amounts proportional to the extent and severity of the infection. If only the sigmoid and rectum are involved blood and pus may be frequently evacuated and yet the patient complain of constipation because the stools may be scybalous. Evacuation may be associated with cramps and tenesmus, but the stools as a rule are not watery. A septic temperature occurs in acute cases and a low grade fever is commonly found in acute exacerbations of long-standing disease. The patient loses weight, develops a secondary anaemia and chronic invalidism results. As in other chronic infections achylia is frequently found. This should be recognized and corrected because it in itself frequently causes diarrhoea and the colon is in no condition to cope with added insults.

Complications of the disease include severe anaemia which at times require transfusion, and peri-rectal infection, necessitating drainage. Malignancy superimposed on the colitis is seen rather frequently, but profuse haemorrhage is rare.

Treatments advocated for ulcerative colitis have been as numerous and varied as can be imagined. Irrigations from above and below have been done with every antiseptic, astringent and soothing lotion that could be safely employed. Bland diets, drugs without number have been administered orally, intravenously and intramuscularly. In a limited time we hope to see treatment standardized. Autogenous vaccines or filtrates should be prepared as early as possible and all distant foci removed at such time as seems discreet. Achylia if present should be treated with HCl in the usual way and tincture of iodine by mouth seems to be of considerable value. Gentian violet would appear to be a reasonable drug to use as in rather low dilutions it kills the diplococcus in the test tube. Irrigation of the colon is really of little value and in some patients aggravates the spasm and tenesmus. In acute cases with high fever Bargen has used immunized horse serum with amazing results. The serum as yet is not available for general use, but in time will doubtless be routinely used for the acutely ill patient.

The diet should be liberal and well balanced, but containing as little roughage as possible. Frequent sigmoidoscopic examinations in the acutely ill patient unwise, but in the chronic type of case with secondary infection, local applications through the sigmoidoscope are of considerable assistance. The drugs of most value for this purpose are Gentian violet, mercurochrome, witch-hazel, sodium chloride solution and argyrol. For oral administration, cod-liver oil, Blaud's pill, tincture of iodine, kaolin and bismuth are indicated in selected cases. Transfusion should be done when indicated.

EMPYEMA.

By R. H. Meredith, 2T8, and M. M. R. Hall, 2T8.

The study of empyema has received an enormous stimulus during the last few years, partly because of its frequency as a complication of wounds in the chest during the war, but chiefly because of the extraordinary number of cases which developed in the course of recent epidemics of measles, streptococcic pneumonia and influenza. As a result of this study much light has been thrown on the pathology of the condition, on the physiological mechanics of the thorax and on methods of treatment. Empyema is a collection of pus in the pleural cavity. The effusion may be purulent from the beginning, but more frequently it is at first serous or serofibrinous, becoming purulent later. The disease is commonest in the first two years of life, and the mortality is highest during this period. Choyce states that 45% occur in children under 10. Boyd states that two-thirds of all cases occur in males.

Etiology.

The causative agent during the influenza epidemic was most commonly the streptococcus, while in peace time the pneumocccus is responsible for the majority of cases. It is of considerable importance to recognize which of these organisms is responsible because of the difference in treatment. Tuberculosis is given as the cause of 2% of cases by Wilensky, and of 10% by Choyce. Other organisms as staphylococcus and B. Coli are more rare; in the latter case one may be able to detect a fecal odour from the pus. When the fluid aspirated is found to be sterile, T.B. is the likely cause.

The infection may reach the pleural cavity in three ways:

1. By spread from neighboring organs.
2. By introduction from without.
3. By the blood stream.

1. By spread from neighboring organs.

The great majority of cases are due to the spread of infection from a pneumonic lung. Two great types must be recognized: Those which follow a lobar pneumonia and are caused by the pneumococcus, and those which accompany the streptococcal pneumonias so frequently associated with influenza, and less commonly with measles and the other exanthemata. The distinction is fundamental since it has an important bearing on treatment. Thus if the chest is opened in the early stages of non-streptococcal empyema, the result may be disastrous since the pneumonia is in full bloom, the patient's vital capacity is already perilously low, he is cyanosed and suffering from marked air hunger, and even a small opening in the chest wall may cause collapse of both lungs and death. In pneumococcal empyema, on the other hand, the presence of pus is usually not recognized until the pneumonia has subsided, so that the condition of affairs is similar to that obtained by delay of open drainage in streptococcus cases.

As to the method of infection of the pleural cavity from the lung it has been shown that in the streptococcus cases it was nearly always by rupture of a small sub-pleural abscess, the same holding good also for at least a fair number of pneumococcus cases. This perforation may be small, when it will probably heal readily, but not infrequently it remains open, and if it communicates with a bronchus we have the production of a pleuro-bronchial fistula which is one of the causes of the empyema becoming chronic.

The infection may come from other neighboring organs than the lung as from an abscess of the mediastinum, or of the chest wall, a subphrenic abscess, a liver abscess or suppuration of the gall bladder.

2. By introduction from without.

A perforating wound of the chest is liable to be followed by empyema, and this was of considerable importance in war surgery.

3. By the blood stream.

In rare cases empyema may be apparently a primary condition and these cases are undoubtedly due to blood stream infection. More frequently it can be traced to some focus as in tonsilitis, appendicitis, acute otitis media, or puerperal sepsis. An empyema may develop in the course of a bacteremia, but in this case it acts as a fixation process, and the prognosis is more favorable.

Pathology.

An empyema, especially when secondary to a lung infection, is usually preceded by a slight exudation of serous fluid which later becomes purulent. An abundant inflammatory exudate is deposited on the surface of the lung and on the parietal pleura, and pus is poured into the intervening space. So massive is this exudate that there is a tremendous loss of body nitrogen, a point of considerable importance in treatment, a diet of high caloric value being indicated. The usual site of a subpleural abscess is the convex surface of the lung, giving rise to a diffuse empyema. If the abscess is located in a fissure there will be an interlobar empyema. If it is on the medial aspect of the lung, one or more pockets of pus will form retrosternally between the lung and the mediastinal pleura.

The inflammatory exudate consists mainly of a dense network of fibrin in the meshes of which are serum and large numbers of polymorphs. Later the exudate is invaded by fibroblasts which lay down bundles of collagen fibres, so that eventually the lung is covered by a layer of exceedingly dense fibrous tissue which may be an inch in thickness. A similar deposit is laid down on the parietal pleura and may plug the exploring needle. This exudate may play a large part in preventing expansion of the lung after the pus has been evacuated. One of the great advantages of Dakin's solution for irrigation of empyema is that it exerts a decorticating influence by loosening this deposit and bringing it away in fragments, thus facilitating the expansion of the lung. This deposit also causes fixation of the mediastinum so that when the affected side is opened the mediastinum does not move over, causing collapse of the other lung as it will do in a normal chest, or if an empyema is opened too early. Thus pneumothorax is dangerous and frequently fatal in early cases of empyema, whereas in the later stage it may be of value. The exudate causes adhesions of the lung to parietal pleura so that the pus is encapsulated and the lesion takes on the character of a local abscess.

The pus varies with the infecting organisms. In pneumococcus cases it is thick, creamy and yellowish or green in colour. When due to streptococcus is usually but not always, thinner and more watery and contains flakes of fibrin. Tuberculous pus is also thin and contains masses of caseous material. A fecal odour suggests B. coli infection.

The effect on the lung of the purulent exudate is of great importance. The lung is lifted up on the surface of the fluid, and becomes more and more collapsed, until it may form a small compressed mass at the hilus. Moreover a purulent material exerts a much more deleterious effect on the lung substance than does serous fluid. There may be necrosis and destruction of the lung tissue at the seat of pressure, together with an interstitial inflammation, resulting in fibrosis and contraction, and thus still further interfering with subsequent expansion of the lung.

Signs and Symptoms.

The onset may be insidious especially when complicating a severe illness as pneumonia, or following a simple pleurisy; then only the maintenace of fever, the absence of a crisis, and the persistence of the physical signs in the chest

and of constitutional symptoms suggest some suppurative complication. In some cases epigastric pain, tenderness, and vomiting are the earliest symptoms. Fever is variable; it may be slightly irregular with occasional sharp rises, or there may be a rigor with persistent high temperature. There is often a constant hard cough which greatly aggravates the pain. Dyspnoea is an indication of the rapidity of formation and amount of the effusion. The physical signs are those of fluid in the chest and vary with the degree of compression of the lung. In chronic cases, however, marked dullness associated with a small empyema is due to the great thickening of the pleura; the breath sounds are then faint and tubular. The change is physical signs of an empyema following immediately after a pneumonia may be very slight.

Course.

If an empyema is left alone it may be recovered from provided it is of limited extent. It may kill the patient from septicemia, amyloid disease may develop, or the empyema may rupture externally or into a neighboring cavity such as a bronchus, the oesophagus, or the peritoneal cavity. That an empyema may be followed by a metastatic abscess in the brain is well known. Such an abscess, however, must in every case be due to the abscess in the lung which gave rise to the empyema, for there is no conceivable route by which an embolus could pass from the pleural cavity to the brain.

Following an operation, the empyema may heal fairly rapidly with expansion of the lung and obliteration of the cavity. Very frequently, however, the condition becomes chronic, pus continues to discharge from the opening for months, or it may be years, and the lung refuses to expand. For this there may be a number of reasons, but the most important are the continuance of the infection and the encapsulation of the lung by the dense fibrous deposit. The former is frequently due to the persistence of a pleuro-pulmonary fistula through which fresh infection is continually occurring. It is for this reason that the method of treatment by irrigation with Dakin's solution is often so effective, for the sodium hypochlorite both sterilizes the cavity and decorticates the lung by dissolving the exudate. The greater the chronicity the thicker the exudate becomes. In old cases there may be calcification of the pleura.

Treatment.

The epidemic of haemolytic streptococcic infections of the respiratory tract associated with empyema which ravaged the military camps of the U. S. during the winter of 1917-18 did much to clarify the general understanding of not only the pathology, but also the treatment of empyema.

The mortality rate was 30.2%. Because of this an empyema commission of the U. S. army was formed. Evarts Graham, from whose monograph much of the material in this paper is taken, was a member of this commission.

As a result of the findings of this commission the mortality rate was reduced to 4.3%.

The principle of early open drainage in conditions of acute empyema was in vogue when the high mortality rate was obtained. This tendency was probably largely due to the feeling that the treatment of acute inflammations elsewhere. It ignored the conditions which have been shown by animal experimentation, by theoretical deductions, and clinical findings, which make open drainage in acute empyema a positively harmful procedure which must often be in itself an immediate cause of death.

Open pneumothorax means a condition in which there is free entrance and exit of air into the pleural cavity through an opening in the chest wall. It used to be thought that when pneumothorax was done the lung on the affected side collapsed and the other lung carried on the work of respiration. That is, the mediastinum was looked on as being rigid, but, in reality any altera-

tion of pressure in one pleural cavity is accompanied by an alteration of pressure to practically the same extent in the other pleural cavity, provided there are no adhesions of the lung to the parietal pleura or no thickening of the structures of the mediastinum. Thus both lungs must function although both are compressed.

Pneumococcic empyema is almost always a complication of lobar pneumonia, and it usually is not recognized until after the acute stage of the pneumonic process has subsided, and at a time when probably the disappearance of much of the exudate from within the lung has increased the vital capacity. On the contrary, in the haemolytic streptococcic empyema it is customary for a massive pleural effusion to occure early in the course of the disease, and thus cyanosis and dyspnoea are prominent much more than in the pneumococcic variety. Open pneumothorax throws an additional burden on respiration and adds greatly to the danger. Another effect of open pneumothorax is in the great loss of body heat, this having been shown to be greater than if the intestinal contents are left lying on the abdomen for a corresponding length of time in doing a laparotomy. There is also the risk of secondary infection, and in failure of an already damaged heart.

In the treatment of acute empyema, open pneumothorax should not be used. Drainage by aspiration is the most satisfactory treatment in the acute stage. The explanation of the relative freedom from systemic effects of these retained accumulations of pus is to be found in the comparatively slight absorption of toxic material through the dense fibrinous exudate which surrounds and limits the abscess. Absorption depends on pressure and surface exposed. Thus absorption can be prevented by aspiration, and the time to aspirate can be determined by the toxicity as shown by the pulse and temperature. A small number of cases may be completely cured by aspiration. But (1) if one does not seem to be making progress by aspiration or (2) if aspiration for three weeks does not clear up the condition, or (3) if one finds frank pus, resort should be made to operative procedures. At this time the vital capacity is improved, the abscess is walled off, and the condition of the patient is much better than in the acute stage.

At this stage one has the choice of intercostal drainage or rib resection. Under ordinary circumstances the latter would afford better drainage, but it has certain disadvantages: (1) There is a slight chance of an open pneumothorax being produced by the breaking down of adhesions. (2) Infection of the cut ends of the ribs may occur with resultant osteomyelitis, being one of the causes of chronic empyema. This may be minimized by rubbing bone wax into the open ends of the rib at operation. (3) Small fragments of bone may fall into the cavity at time of operation or be separated off as sequestra later giving rise to chronic sinuses. Therefore it is thought best to use intercostal drainage, unless the space between the ribs is too small as in infants or rarely in adults. If better drainage is desired later the opening may be enlarged by the resection of one or more ribs. Either operation may be done under local anaesthesia.

In doing the operation of intercostal drainage the pus is located by percussion and an aspirating needle is put into the abscess, usually in the ninth space in the mid or post-axillary line. The opening for the tube is made by cutting along the needle, parallel to the ribs. The tube is inserted, the dressing packed around it with adhesive to make it air-tight and suction drainage applied as with a Sprengel pump.

The object of treatment is to sterilze and obliterate the cavity, and with the above treatment healing usually takes place in five to eight weeks. If this does not occur irrigations twice daily with Dakin's solution may be used. Blow

bottles are of great value in assisting the expansion of the lung and obliteration of the cavity.

The chief cause of failure of the cavities to be obliterated is inability of the lung to expand properly due to two factors: (1) fibrosis of the lung and (2) the presence of a thick inelastic fibrinous exudate covering the exposed surface of the lung. The great advantage of Dakin's solution is illustrated in the latter cases since it both causes sterilization of the cavity and decortication of the exudate, greatly reducing the number of cases requiring surgical means of obliterating the cavity.

Among the operations employed for causing obliteration of the cavity are: (1) Fowler-Delorme for removal of the fibrinous exudate, and (2) Estlander or Schede types of Thoracoplasty with removal of several ribs, allowing the chest wall to fall in. In both types of operation the mortality is high, and in both only one of the two objects of treatment is carried out, namely, obliteration of the cavity. Moreover, collapsing operations give a poor cosmetic result, do not always result in healing, and reduce the vital capacity to about half, leaving the patient in a condition of decided danger in case of subsequent respiratory infection.

As mentioned before, careful attention to the nutrition of the patient is as important as any other phase in the treatment of empyema. Because, unless special attention is paid to diet, there will be a daily negative nitrogen balance with marked emaciation. The patient should be given 3,000-3,500 calories per day. When the temperature is raised and the appetite poor they may be given fruit drinks containing glucose or lactose, or 5% glucose intravenously.

Finally, after the wound is entirely healed, the patient should be carefully watched for at least six months, to be sure there is no recurrence.

SEDIMENTATION IN PULMONARY TUBERCULOSIS.

By R. W. McNeely, M.D.

In presenting a short resume of results of Sedimentation Tests recently made at the Mountain Sanatorium, attention is directed to a modern application of a very old principle well known to physicians even in the time of Galen.

In 1771 Hewson observed that increased rate of settling of blood corpuscles, leaving the clear serum above with the formation of the "crusta phlogistica," was a sign of inflammation.

In citrated or oxalated blood the erythrocytes settle with varying rates in diverse physiological and pathological conditions. From a review of the different theories explaining this phenomenon one infers that augmentation of plasma globulins and fibrinozen are the principal factors in increased sedimentation rate (SR) while surface tension, capillary attraction, hemoglobin content and size of erythrocytes, electrical reaction and the presence of antibodies may influence the reaction.

The rate varies greatly in different animals. Only thirty minutes is required for the sedimentation of the blood from horses, while with cows' nearly a week's time is necessary for complete sedimentation.

The cells of defibrinated blood require a much greater period of time to sedimentate than the cells of citrated blood from the same person. The rate is increased in pregnancy, possibly owing to a greater absorption of globulin than usual and the test has been used in the differential diagnosis between fibroids and pregnancy and also in estimating quiescence or activity in pus tubes. Very high figures are frequently

found in lobar pneumonia, syphilis, arteriosclerosis, polyarthritis and in acute infections accompanied by high temperature. There is a definite increase in active tuberculosis depending apparently on the pathological changes taking place in the lungs. Of outstanding importance, from a practical viewpoint, is Cutler's observation that increased sedimentation depends on the degree of cellular activity going on in the body and reflects the disturbance produced in the organism through the absorption of the products of infection.

In 1894 Prof. Biernacki published several papers on the subject. Fahraeus wrote a monograph on sedimentation in 1918 and more recently Westergren has done some very interesting work and introduced simplification of technique. Friedlander has also made valuable contributions more recently. An account of Westergren's method will be found in the July, 1926, number of the American Review of Tuberculosis and an excellent article by W. H. Morriss in the December, 1924, number of the same journal. The August, 1926, number of the Journal of Laboratory and Clinical Medicine also contains an interesting article by Morriss and Rubin.

From a perusal of the numerous articles published in recent years on the subject, one notes that there are as many methods of sedimentation determination as there are workers in this branch of research—all of them excellent—but in the interpretation of results it is difficult to decide what is a normal rate for a normal individual. There has been no standardization, either of method or of reporting results, thus detracting from the undoubted practical value of this important test. Many different methods were tried out in a series of preliminary tests at the Sanatorium, and an attempt was made to evolve a technique, simple, practical, accurate and available alike for the laboratory technician and for the practicing physician—especially for the physician interested in tuberculosis.

For the purposes of this article, the sole object of which is to stimulate interest in the subject and to provoke constructive criticism, rather than to summarize immature conclusions, we believe that our purpose will be best accomplished by describing in detail the technique which we believe embodies the salient points and eliminates the innacuracies of micro methods. We have not tried out the Sedio-Tassometer, the use of which is highly commended among the micro methods, as, so far as we are aware, none of these instruments has yet been imported here, though one is on order and will be available for trial in the near future.

Technique of Sedimentation Tests in Use at the Mountain Sanatorium.

Requirements.

1. Record Syringe, 2 cc. capacity, with luer tip.
2. Bottle of sterile, three per cent. sodium citrate, in normal saline.
3. Small storage tube (as used for Wassermann).
4. Sedimentation Pipette (Exax 1 cc. graduated in hundredths).
5. Suitable pipette stand to insure absolute perpendicularity.

In storage tube place 0.6c.c. of sterile sodium citrate solution. The syringe and needle being washed out with citrate will prevent any coagulation when blood is drawn into the syringe. In disinfecting the skin ether or alcohol should be applied with sterile gauze in preference to cotton. The tourniquet is applied to arm and 1.4 cc. of blood is quickly withdrawn. Prolonged stasis should be avoided.

The blood is expelled immediately into storage tube and thoroughly mixed by inverting the tube several times, holding the dry finger over the mouth of the tube.

When ready to commence the test the sedimentation pipette should either be perfectly dry or citrate solution may be first drawn through and then expelled. Draw up the blood into the tube several times and allow to run

back. Do not blow the blood back or bubbles will be formed. Then draw up the blood to the zero mark, place in rack, inserting the bottom of the tube in plasticene before withdrawing the finger from the upper end. This dispenses with the necessity of rubber corks or pressure to insure non-leakage.

Readings are taken every ten minutes for one hour and sedimentation rate (SR) reported in percentage, at the end of the hour. Other readings may be made at two hours and at twenty-four hours if desired, but for our requirements we found the hour period of greatest interest. If desired, the amount of "crusta phlogistica" may be read off in percentage at the end of twenty-four hours. In reading percentage of clear serum above the settled erythrocytes the layer of white blood cells is included.

The syringe is then washed in tap water, alcohol, ether and sterile water. Needles are boiled and before beginning another test citrate is drawn into syringe and then expelled. If desired, 0.6 c.c. of citrate may be drawn into syringe and the blood drawn from the vein up to the 2 cc. mark and mixing may be done in the syringe, but the technique as outlined previously has been found very satisfactory, especially when working with children or nervous patients.

Room temperature is satisfactory. The citrated blood may be kept several hours before the test is made without modifying the results. If any clotting occurs sample should be rejected. Imperfect sedimentation takes place if tubes are not thoroughly cleansed.

After making the test the sedimentation tubes are withdrawn from the rack holding finger over the upper end, otherwise leakage will occur as the plasticene should not stick to the lower end of the tube. The blood runs out easily and running water should be run through the tube till thoroughly clean. Then tubes are sterilized by boiling and are ready for use after removal of moisture.

Graphs are made recording the progress of the curve every ten minutes and percentage recorded. The graph is filed with chart, for comparison with future tests. The normal graph approaches the horizontal, while a graph showing marked perpendicularity indicates activity. A flattening out of the graph from the perpendicular towards the horizontal indicates improvement in condition.

Satisfactory sedimentation pipettes are now being supplied by Ingram and Bell, made by the Exax firm according to our specification. These tubes are 13¾ inches long or 350 m.m. The capacity is 1 cc. and graduations are in hundredths. The zero mark is 8 ins., or 205 m.m., from the base, and the graduation is carried within 5 m.m. of bottom.

To insure absolute uniformity in bore, we test all our pipettes with citrated blood of one person, taking readings at one, two and twenty-four hours. If there is any deviation the pipettes are rejected. After using pipettes, if any plasticene should adhere to the bottom, it may be easily removed by washing in ether.

Being convinced of the importance of the S.R. after following a series of cases for over a year, we decided to apply the test to a larger number of patients, and now a routine test is being made on each patient who enters the Sanatorium. During the past month two hundred and fourteen tests were made. Of these seventy-four were on children in the Preventorium and one hundred and forty on adult patients in the Sanatorium. Of the children, thirty nine were girls and thirty-five boys. Of the adults, thirty-two were men and one hundred and eight were women.

We deprecate the tendency noticable in some non-Canadian journals to attempt to express a percentage in a series of one, two or three cases, instead of in one, two or three thousand—so kindly bear in mind that the figures we quote will undoubtedly be modified

to some extent when other Sanatoriums take up this work.

Forty-four and six-tenths percent of our Preventorium Children had a SR of three percent or less.

Eleven and four-tenths percent of adults had a rate of three percent or less. Examine these figures from every angle and one tremendous fact stands out. More Preventoriums are needed. Prevention is more rational, more economical, more humane than attempted cure.

Twenty and three-tenths percent of the children had a rate of six percent or over, while seventy and nine-tenths percent of the adults had a rate of six percent or over.

Five percent of the children had a rate of ten percent or over, while fifty-one and four-tenths percent of the adults were over ten percent. Twenty-seven and one-tenth percent of adults were over twenty percent and eleven and four-tenths percent of adults had a rate of over thirty percent. One boy with bilateral pleurisy with effusion had a rate of thirty-three percent.

We think that the use of a percentage basis of estimation of the S R has decided advantages. Readings are instantly made and the amount of clear serum, the percentage volume of cells overlying the layer of erythrocytes and the percentage volume of the settled red cells when sedimentation is complete can readily be computed.

Our interest in this preliminary work has been primarily the S R during the first hour, but there is a vast amount of interesting data to be compiled, such as the effect of drug therapy, artificial pneumothorax, phrenicotomy, thoracoplasty, etc., on the S R, and it is only by the combined efforts of many researchers working along definite lines with a standardized technique that such data can be obtained.

In recent years the S R is attaining greater and greater importance as a clinical re-action in the progress of tuberculosis. Generally speaking, patients who have a low S R are regarde as having a much better prognosis than those with a high rate. In fact, a very high S R is an indication for the exhaustion of the attending physician's resources in an effort to control the devastating cell destruction taking place in the body of his patient and by repeating the tests at suitable intervals valuable knowledge of therapeutic effects and of progress of the disease is obtained. One of the most cheering experiences in our work during the past year has been to note the straightening out of the sedimentation graph in certain instances, and in many cases a high S R has proved a valuable additional factor to other clinical findings, prompting the initiation of calcium chloride therapy, pneumothorax treatment, curtailment of exercise, etc. Certain workers believe that a diminution of the S R precedes by several weeks manifest clinical improvement.

In patients with bone tuberculosis and discharging sinuses we found a high sedimentation rate, but sufficient work has not yet been done to draw any definite conclusions. We trust that more work will be done during the present summer, so that accurate data may be available.

Westergren has observed that there is an inhibiting influence on the sedimentation rate in cardiac decompensation. In the case of a boy of fourteen we had a striking confirmation of this—his S R being the lowest in our whole series of cases.

In order to give some general idea of what to expect in making sedimentation readings we have appended a table taken from our series of readings to illustrate the differences in rate in minimal active cases, moderately advanced, far advanced, active and arrested. Considerable variation from these figures is, however, to be expected, and we have as yet not formulated any definite rate for the various classifications.

As a tentative programme for those

interested in sedimentation work during the coming year might we suggest the following lines of investigation:

(1) The effect of pneumothorax on the S R.

(2) The effect of intravenous calcium chloride therapy for a period of three to six months on the S R in cases of pulmonary tuberculosis.

(3) The effect of heliotherapy in children and adults on the S R.

(4) What is the normal S R rate in Medical students entering their first year at the University?

In conclusion, we can at least assure these students who take up the sedimentation test as a problem for investigation that they are going to find it intensely interesting, and we believe that they are going to do more intelligent and better work from their efforts to find the REASON.

SEDIMENTATION RATE IN VARIOUS SANITORIUM PATIENTS.

No.	Sex.	Classification.	Sedimentation rate in percent. at 10 min. intervals. 10 m.	20 m.	30 m.	40 m.	50 m.	60 m.	S.R.	PROGNOSIS.
1.	Male.	Minimal.	0.1	0.2	1	1	1.5	2	2%	Excellent
2.	"	Minimal.	0.2	0.75	1	1	1.2	1.7	1.7%	Excellent.
3.	"	Minimal.	0.25	0.5	1	1.5	2	2.5	2.5%	Excellent
4.	"	Mod. advanced, active..	0.2	1.5	2	2.5	3	3.5	3.5%	Good.
5.	"	Far advanced, active..	2.5	9	19	27	32	37	37%	Poor.
6.	"	Far advanced, active...	2	10	21	30	39	46	46%	Poor.
7.	Female.	Far advanced, active...	1	8	12	20	22	25	25%	Poor.
8.	"	Far advanced, arrested.	0.5	0.7	1	1	1.2	1.5	1.5%	Excellent.
9.	"	Far advanced, active...	2	6	12	19	28	33	33%	Poor.
10.	"	Far advanced, arrested.	0.2	1.5	4	5	6	7.5	7.5%	Good.
11.	"	Mod. advanced, arrested	0.2	0.3	0.4	1	1.5	2	2%	Good. Discharged.
12.	"	Mod.advanced, quiescent	0.5	1.5	3	4	5	6	6%	Good; on exercise.
13.	"	Bronchiectasis	0	0	0.2	0.5	0.75	1	1%	Good.
14.	"	Mod.advanced, quiescent	0.2	0.5	1	1.3	1.5	2	2%	Good; on exercise.
15.	"	Mod. advanced, active..	0.5	1	2	3	4	4.5	4.5%	Good.
16.	"	Non-tuberculous	0.3	0.5	0.6	1	2	3	3%	Good.
17.	"	(Child) far advanced...	0.3	4	5	7	9	12	12%	Doubtful.
18.	Male.	Bronchiectasis and Lues	2	5	10	13	17	22	22%	Poor.
19.	Female.	Far adv. Pulm'ry T.B. Quiescent and Tuberculous knee, active..	6	15	26	35	45	52	52%	Doubtful
20.	"	Far adv. Pulm'ry T.B. Quiescent & Potts Dis.	2	4	8	13	18	22	22%	Fair.

PYELITIS OF PREGNANCY.

R. C. Montgomery, M.B., B.Sc. (Med.)

The so-called pyelitis of pregnancy was first mentioned in the literature in 1841 by Rayer of Paris, who said: "The enlargement of the uterus in pregnancy is the frequent cause of cystitis, and in consequence of this, inflammation of the ureters, the renal pelves, and of the kidney itself." It was not until 1910 that detailed clinical study was made of this condition. Now it is evident that there is more involved than the pelvis of the kidney, that there is retention of urine in the whole urinary tract, with consequent distention of the ureters and of the pelvis of the kidney. In 1924 it was shown by McClelland to occur most often in primiparae during the middle three months of pregnancy, and is most common in young women under twenty-five years of age.

Although many bacteria have been found in this condition the colon bacillus is undoubtedly the most prominent. Kidd, of London, found the colon

bacillus in 117 out of 140 cases. Streptococcus, staphylococcus, and gonococcus have all been isolated. Rosenow thinks it is likely that the primary invader causing the infection is either staphylococcus or streptococcus. After the kidney resistance is lowered the colon bacillus grows so rapidly that the primary invader is overcome. Baker in a study of 50 cases finds the tonsils, teeth and sinuses to be the seat of infection in over 80 per cent. of his cases. Any obstructive lesion of the urinary tract plus any pyogenic focus must be rated as a predisposing cause.

It is interesting to note that Koenter and Neumann claim that there is a lowering of the opsonic index in pregnant women to the tubercle bacillus. Sanno states that he has found evidence of lowered resistance to infection during the last half of pregnancy. Hoffbauer finds lowered opsonic index of the serum to B. Coli in 7 out of 55 cases.

Previous illnesses do not seem to play a very important part, but if the affection persists in the puerperium it is probable that an obstruction was present before pregnancy began. Pugh believes that pyelitis in children is an etiological factor in the production of ureteral stricture, with pyelitis of pregnancy later on. It would seem that many so-called cases of pyelitis of pregnancy cure themselves spontaneously, while many severe ones are wrongly diagnosed.

Three views are commonly held as to the point of entrance of pathogenic bacteria. First, an ascending infection from the lower urinary tract. Second, lymphogenous. Third, hematogenous.

Recent work has shown that the most important factor is urinary stasis. This was first noticed by Cruvelhier, who performed autopsies on pregnant women. He found dilated ureters to be more common on the right side. The cause of this stasis it not entirely clear. Most authorities consider that it is not due to pressure by the pregnant uterus. Opitz believes it to be due to a sharp kink in the right ureter as it crosses the right iliac artery.

Franz believes that there is some obstruction at the pelvic brim, as the ureters are not dilated below this point. There is a predisposing cause present in the general pelvic congestion, but the fact remains that the ureter is not dilated right down to the bladder. Possibly there is obstruction of some kind as the ureter passes in the region of the broad ligament. A recent paper by Hoffbauer shows certain anatomical conditions in the juxtavescial portion of the ureter and trigonum vesicae. The lumen of the lower part of the pelvic portion of the ureter is narrowed by hypertrophy of the musculature and hypertrophy of the connective tissue. The constriction is accentuated by a ring produced by hypertrophy of the ureteral sheath. Similar hypertrophy is found in the trigonum, being present in the longitudinal fibres which are continuous with the ureteral sheath. He notes that the distal end of the ureteral dilatation lies at the level of the parametrium and associated with this is demonstrable delay in the ureterine action. Yet there is very little obstruction met with when the ureters are catheterized. As it is a well known fact that the pregnant uterus is rotated to the right, Swalbe believes that there is a similar rotation on the part of the pelvic ureter in relation to the abdominal ureter. Hoffbauer believes that the close association of the lower uterine segment and the base of the bladder produces rotation of the latter as the uterus moves to the right. As the muscle hypertrophy stiffens the trigonum vesicae and pelvic ureters, a kink is produced in the right ureter, and a stretching in the left.

Symptoms: There appears to be two distinct types of infection, the symptoms depending on the amount of interference with the pelvic drainage. No doubt some with very little obstruction pass undiagnosed. The acute type begins with chills, nausea, vomiting, and a sharp pain, usually on the right side

which is worse when the patient takes a deep breath. The other type shows only a slight rise in temperature associated with a dull apathetic state. One notable feature is the slowness of the pulse, which is quite out of proportion to the rise of temperature. McClelland, reporting on thirteen cases, tabulated the symptoms in order of frequency, as follows:

	Cases.	Percentage
Pain	13	100
Frequency	10	77
Burning	7	54
Chills with fever..	7	54
Vomiting	3	23
Hematuria	1	8
Blurred vision ...	1	8

Examination: Tenderness in the costo-muscular angle of the affected side is always found, although the kidney may not be palpable. Some cases show marked abdominal rigidity, others show none. Normally deep palpation shows tenderness along the course of the ureter. All cases show pus in the urine, most show some albumen. The white blood count varies from 10,000 to 25,000. Blood pressure usually is unchanged. Temperature varies from 99 to 104 degrees.

The picture as seen through the cystoscope is one of general redness, being more marked in the region of the affected ureter. Many of the cases show cloudy urine flowing from the ureter, and the ureteral catheter seldom meets with any obstruction. One striking point is the free flow of urine from the catheter as if a pocket of urine is tapped. Very often this procedure gives immediate relief of pain.

The history of pain usually in the right flank, associated with tenderness in the costo-muscular angle, frequency of urination with burning, would at once suggest some lesion of the urinary tract. A catheter specimen of urine containing pus would complete the picture. However, the urine should be cultured in all cases and if no Bacilli coli are found a routine examination should be made for the tubercle bacillus.

A previous history of renal colic and the X-ray will rule out any calculus. Right-sided pain, low temperature and slow pulse often suggests appendicitis, but a catheter specimen of urine will usually make the diagnosis clear.

Treatment: As it is generally believed now that the stasis of the urine is one of the most important factors in this condition it would seem that any treatment should be directed in a way to correct this lesion.

It is suggested recently that pituitrin be used in an effort to stimulate ureteric peristalsis, and in this way overcome any dilatation.

Usually urinary antiseptics are tried especially if the case is a mild one. The patient is instructed to drink as much water as possible — a glassful every hour. With this is given Potassium Citrate and Potassium Carbonate of each sixty grains per hour. Recently Hexyl Resorcinal 0.15 gms. every four hours is used. With this drug the water intake is limited to 50 ounces in 24 hours.

Pride also advises the knee-chest position for ten minutes three times daily, on the assumption that the kidney is displaced downwards, producing a kink in the ureter.

If the patient is acutely ill or does not respond to the above treatment, ureteral catheterization is advised. There is a certain amount of discomfort to the patient in this procedure, but the whole operation can be done under local anaesthesia with the aid of sedatives. Sometimes drainage is established in twenty-four hours, at other times the catheter must be left in for weeks. This procedure seldom brings on labour unless the patient is at full term.

Pyelitis occurs in the puerperium and usually responds very well to urinary antiseptics However, a recent paper by Corbus and Danforth shows a definite obstruction in the urinary tract in a series of 13 cases. There is

apparently some lesion present before pregnancy. Such cases demand adequate drainage if serious permanent damage to the kidney is to be avoided.

Prognosis: In a recent article by Hoffbauer, a thorough study was made of twenty-seven patients who had pyelitis during pregnancy. The length of time which had elapsed between labour and the remission of the condition existing in the urinary tract varied from 4 to 33 months.

Conclusions: The conclusions were as follows—

1. Out of the 27 cases examined, 9 had had the pyelitis during the puerperium. There was no persistent dilatation of the ureter noted on re-examination, nor were any of the previously existing bacteria found.

2. Bacillus coli had been cultured from the urine during pregnancy in 14 out of 18 cases of pyelitis. On re-examination, the same bacteria were found in renal pelves of 7 cases.

3. When signs and symptoms of pyelitis had developed during the last 10 days of pregnancy, no after-effects could be demonstrated in the urinary tract.

4. A marked dilatation of the ureter was found in 8 of the patients examined.

5. When dilatation was present it involved the upper half of the pelvis, as well as the abdominal portion of the ureter.

6. The histories of these cases demonstrated a correlation between the severity of the symptoms and the persistence of ureteral dilatation later on.

7. Albuminuria persisted in one case for a year.

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CORONARY THROMBOSIS.

A summary of an article by Drs. John Parkinson and D. E. Bedford, appearing in The Lancet of January 9th. 1928.

By E. Arthur Cummings 2T8.

When a man of advancing years is seized with severe pain across the sternum which continues for hours and is accompanied by shock, collapse and dyspnoea, he has an anginal attack of no ordinary kind.

Coronary thrombosis is a complication of arterial disease which can be compared with cerebral thrombosis. Up until a few years ago it was regarded as a terminal complication of angina pectoris. Now it is a well recognized clinical syndrome, and is easily diagnosed during life. The recognition of coronary thrombosis is of importance clinically because it has been mistaken for abdominal conditions and laparotomies have been performed; mistaken also for angina pectoris, the treatment of which is entirely different.

This paper is based on a clinical and pathological study of one hundred cases.

Pathology.

In the vast majority of cases the pathological basis of cardiac infarction is atheroma of the coronary arteries. Final occlusion is usually due to thrombosis at the site of constriction or ulcer-

ation of the vessels. In 70 per cent. of the cases of recent infarct due to atheroma, thrombosis was visible to the naked eye. Less than 10 per cent. of the cases of all cardiac infarcts are due to closure of the mouths of the coronary arteries by syphilitic disease, with or without thrombosis. The most common site for coronary thrombosis was the left anterior descending branch in the proximal three centimeters of its course.

Size and distribution of the infarct: The area of infarction may vary from the size of a quarter to an area involving the whole of one ventricle. The site of an infarct depends on the area of muscle supplied by the occluded artery. If the left anterior descending coronary is occluded, the apex, anterior wall of the left ventricle, and the anterior part of the interventricular septum may be involved. The infarction is always more extensive on the endocardial than on the pericardial side. The first sign is a slight swelling and pallor of the myocardium. Later the area becomes sharply demarcated, and turns a yellow or grey color, with a red injected border. The area may be diffusely mottled with hemorrhage. Later it becomes organized. This process runs a course of six weeks. The immediate cause of death was cardiac failure in 75 per cent. of the cases.

Complications—Rupture of the heart was present in five cases and aneurismal bulging in five.

The Clinical Picture.

Incidence—Coronary thrombosis is much more common in males than in females. The age incidence is chiefly between forty and seventy years.

The immediate results of coronary thrombosis are, shock, anginal pain and symptoms of heart failure. These, of course, vary so that the disease presents three distinct clinical types.

First—*sudden death at onset.* These patients collapse and die suddenly. This may occur without exertion during the ordinary routine of the patient's life.

Second—*Sudden onset with prolonged anginal pain.* This patient may have had angina pectoris for years, or may have had neither pain nor any other indication of cardiac disease. The attack may be preceded by twinges of sternal pain, followed by agonizing pain in the chest, accompanied by shock and collapse. It is not localized and extends more widely over the chest than anginal pain. The pain may radiate to the arms, back or neck. The pain is not usually brought on by exertion, and even occurs while the patient is sleeping or at meals. The patient is restless, contrasted with the marked rigid immobility seen in angina pectoris. With the pain appear the signs of shock and circulatory failure. The expression is anxious. Sweating is present. Vomiting usually occurs. Some degree of cyanosis and dyspnoea is present.

Physical examination reveals little at this stage. The temperature is subnormal. The pulse is small, usually with normal rythm. The blood pressure is low. Signs of heart failure may be present.

The course—the duration of the severe pain is from half an hour to many days. Nitrites give no relief, and in most cases morphia is required. After recovery from the initial shock the temperature rises from 99 to 102 F. Leukocytosis up to 20,000 is common. After this the course varies, the patient dying suddenly at any time, or else having a rapid and favourable convalescence. The pain gradually disappears.

Third—*Dyspnoea and failure without pain.* When infarction supervenes with a case showing pre-existing signs and symptoms of heart failure the clinical picture may be less distinctive. Pain is not a prominent symptom. Dyspnoea is most prominent. A sudden exacerbation of the signs of failure may be the only evidence of infarction.

Special Clinical Features.

Onset—In 33 cases the pain began during sleep. In another 26 it occurred without exertion.

Site of pain—May be over the lower half of the sternum, or even in the epigastrium. It may be widespread over the chest, radiating to the arms, neck and scapula.

Blood pressure—falls in cardiac infarction. If hypertension is present this fall occurs.

Leukocytosis — The common white blood count, after cardiac infarction is from 10,000 to 15,000. Counts from 15,000 to 20,000 occur.

Fever—During the initial stage the temperature is subnormal. There is then a rise to about 99 or 100 F.

Pulse—Ranges from 100-120, and is small.

Complications—(a) Pericarditis may occur over the area of infarction. A pericardial effusion may be present. Pericardial friction is sometimes heard. (b) Embolism of either the pulmonary or the systemic circulation, secondary to thrombosis over the endocardial surface of the infarcted area.

Relation to angina pectoris—Typical previous attacks of angina occurred in 45 cases. Thus more than half of those suffering from coronary thrombosis have never had angina.

Diagnosis.

(a) From angina pectoris — best shown by a table.

(b) From abdominal conditions—Sudden severe epigastric pain with tenderness, rigidity, vomiting and shock, with small pulse may suggest an abdominal condition. Pointing to coronary thrombosis will be found; 1. A history of previous sternal pain, without history of indigestion; 2, Radiation of pain to arms, neck or jaws; 3, Signs of heart failure.

Treatment.

Morphine is the first essential in treatment in doses of gr. ¼-½ subcutaneously as often as necessary. Nitrites are contra-indicated as they lower the blood pressure and induce tachycardia. Digitalis is used as it reduces the pulse rate and helps relieve the cardiac symptoms. Rest in bed for at least a month after all signs and symptoms have disappeared is necessary.

Prognosis.

Some patients who recover from an attack may lead an active life, some must be restricted in their activities, others are complete invalids.

	Angina pectoris.	Cardiac infarction.
Onset	On exertion	Often during rest or sleep.
Site of pain	Sternal, or mid-sternal	Sternal, often lower third.
Attitude	Immobile	Restless, even walk about.
Duration	Minutes	Hours or days.
Shock	Absent	Present.
Dyspnoea	Absent	Often present.
Vomiting	Rare	Common.
Pulse	Unchanged	Small, often rapid.
Temperature	No fever	Fever follows.
Blood Pressure	Normal or rise	Fall.
Heart sounds	Normal	Distant, sometimes gallop or pericardial rub.
Congestive failure	Absent	Commonly follows.
Electrocardiogram	Often abnormal	Usually diagnostic.

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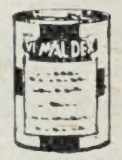

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